Commentary

Oxidative Stress in Diabetes and Periodontitis

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Oxidative stress is an imbalance between the production of a reactive oxygen species and the antioxidant defense, leading to tissue damage. The produced reactive oxygen species, such as superoxide anion, hydroxyl radical, and peroxyl radical result in damage to many biological molecules (including DNA, lipids, and protein), and the prolonged existence of these reactive oxygen species promotes severe tissue damage and cell death.[1,2] It has been proposed that there is a causal relationship between insulin resistance, oxidative stress, and periodontitis and that hyperglycemia is a major factor responsible for the activation of oxidative stress.[1,2]

The presence of malondialdehyde, the products of the hydroxylation of DNA bases such as 8-hydroxy-2′-deoxyguanosine (8-OHdG) were found in crevicular fluid and periodontal pockets.[3] Cell damage is a consequence of the effect of free radicals and chronic oxidative stress.[3] Furthermore, Sawamoto *et al.*,[4] proposed a close relationship between 8-OHdG and periodontal pathogens. When further considering periodontal disease, hypoxia of the tissue occurs at the onset of periodontitis,[5] which stimulates the production of cytokines and inflammatory mediators involved in alveolar bone resorption, including IL-6 and IL-1. With re-oxygenation after hypoxic events, there is an increased level of superoxide anions and other reactive oxygen species, which in turn induce degradation of the NF-kB inhibitor, resulting in greater tissue destruction. Therefore, periodontal disease associated with diabetes, smoking, and/or occlusal trauma leads to periodontal tissue hypoxia and re-oxygenation events, a situation that hypothetically increases the morbidity of periodontal pathology.

Neutrophils in poorly controlled diabetic patients released significantly more superoxide than neutrophils from patients with good glycemic control and from non-diabetic healthy individuals.[6] Neutrophils increased protein kinase C activity, elevated amounts of diacylglycerol, and enhanced nicotinamide adenine dinucleotide phosphate oxidase activity. This suggests that hyperglycemia can lead to neutrophil activation and elevated protein kinase C activity, resulting in increased oxidative stress.[6]

Pendyala *et al.*,[7] demonstrated that increasing oxidative stress can be an important contributing factor in the pathogenesis of diabetes and periodontal disease. Therefore, the co-existence of these conditions could pathologically increase the effect of oxidative stress.

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